

Data Gathering: Biased in Psychosis?

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This study examined whether the probabilistic reasoning bias referred to as a “jumping-to-conclusions” (JTC) style of reasoning, which, according to previous research, is associated with particular psychotic symptoms such as delusions, represents a trait that can also be detected in nonpsychotic relatives of patients with schizophrenia and in nonpsychotic individuals with a high level of psychotic experiences. Participants were, in order of level of psychosis liability, 40 patients with schizophrenia or a schizoaffective disorder, 40 first-degree nonpsychotic relatives, 41 participants from the general population with above average expression of psychotic experiences, and 53 participants from the general population with an average level of psychotic experiences. A “jumping-to-conclusions” bias was assessed using the beads task. A dose-response relationship was found in the association between level of psychosis liability and JTC (defined as needing only a single bead to complete the beads task) (odds ratio [OR] linear trend = 1.59, 95% CI: 1.13–2.24), and, independently, a linear association was apparent between JTC and level of delusional ideation (OR linear trend = 2.59, 95% CI: 1.18–5.69). In addition, the association between psychosis liability and JTC was generally much stronger as the level of delusional ideation was higher. JTC is associated with liability to psychosis (trait), in particular if the psychosis phenotype is characterized by delusional ideation (state).

Key words: cognition disorder/schizophrenia/delusion/
family/neuropsychology

Introduction

Contemporary models of delusions have shown a role for a specific reasoning bias, referred to as a “jumping-to-conclusions” (JTC) style of reasoning, in people with active delusions.^{1,2} Individuals with delusions may incline toward early acceptance and, to a lesser extent, early rejection of hypotheses. More precisely, they show a tendency to seek less information to reach a decision, indicated as a data-gathering bias. This may, under certain conditions, contribute to erroneous inferences and, as is hypothesized, to the formation and/or maintenance of delusions.

The question of whether this reasoning bias has the quality of a state rather than a trait is poorly investigated. In the former case, it concerns a dynamic characteristic that waxes and wanes with the development and remission of a delusional belief. In the latter, it concerns a characteristic that is relatively stable in time, independent of the presence or severity of delusional symptoms. It has been postulated that a JTC reasoning bias with the quality of a state might, at most, be a mediating factor in the *maintenance* of delusional ideation, while as a trait it could possibly contribute to the *formation* of delusions and be part of the vulnerability to psychosis.^{3,4}

JTC was found in 7 out of 8 studies in currently deluded participants, irrespective of a diagnosis of schizophrenia or delusional disorder.^{1,3,5–15} In a longitudinal study the data-gathering bias was found to persist even when the participants were no longer deluded.^{16–18} In another recent study the JTC reasoning bias was found in subjects with an “at risk mental state” for transition to psychosis (without delusions), as well as in subjects in their first episode of psychosis and experiencing delusions of persecution.¹⁹ JTC was also found in a sample of patients with schizophrenia, some of them without delusions, and no correlation was found between JTC and the number and severity of delusions on an “index of deludedness.”¹¹ Taken together, the possibility of a JTC reasoning bias having the qualities of a state remains, but unmistakable evidence for at least partial characteristics of a trait emerges from studies with longitudinal, as well as cross-sectional, designs. The combination of state and trait within one and the same characteristic is not uncommon. For example, the trait low self-esteem is a risk factor for depression, but during

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episodes of depression, low self-esteem will also covary with intensity of the depressed state and contribute to the persistence of depression.^{20–22} More generally, many risk factors for onset of psychiatric disorder are also risk factors for persistence of the same disorder.²³

One way to further investigate the trait and/or state nature of reasoning biases would be to investigate individuals who have no clinical needs but are at risk for psychosis—for example, first-degree relatives of patients with psychosis and individuals in the general population with subclinical psychotic experiences.^{24–28} A number of studies have suggested that the symptoms of psychosis are prevalent in the general population and exist as part of a continuous, albeit skewed, distribution.^{29,30} This suggests that a clinical disorder is at the extreme end of a continuum, which ranges from healthy functioning, through eccentricity and subclinical psychotic experiences, to florid psychosis with clinical need.^{28,29} Not only (sub)clinical symptoms of psychosis but also particular endophenotypic abnormalities in cognition, features of personality, and functional and structural aspects of the brain that have been found in subjects with diagnosis of schizophrenia are found, albeit to lesser degree, in relatives and in individuals at higher risk for schizophrenia.^{31–40}

These findings possibly differentially reflect the expression of a graded genetic predisposition to the disorder, such that relatives, and to a lesser extent individuals with subclinical psychotic experiences, may be more at risk than the general population for later development of schizophrenia or have an undiagnosed but genetically related schizophrenia spectrum disorder.

Two studies have examined JTC reasoning bias in individuals from the general population with a proneness to delusions, both of which showed evidence of such a bias.^{3,4} To our knowledge, no study on JTC reasoning bias involving relatives of patients with schizophrenia has been carried out. In the present study we set out to what degree the JTC reasoning bias as a trait reflects the familial vulnerability for psychosis, to what degree the JTC reasoning bias as a state covaries with delusional states, and whether there is an interaction between psychosis liability and delusions in their effect on JTC. We predicted that the groups with psychosis liability (ie, unrelated controls with a proneness to delusions and, in particular, the nonpsychotic relatives of patients) would display less JTC reasoning bias than patients with a diagnosis of schizophrenia, but more than members of the general population with an average proneness to delusions.^{37,41}

Materials and Methods

Procedure and Sample

Four groups differing in the degree of vulnerability to psychosis were included in the “Cognitive functioning in Psychosis” (CoP) study: (1) patients with history of

nonaffective psychosis, (2) first-degree relatives of patients with nonaffective psychosis, (3) participants scoring high (> 75th percentile) on the positive dimension of psychosis proneness measured by the Community Assessment of Psychic Experiences (CAPE; see Instruments section),^{42,43} and (4) “healthy controls” (ie, participants who scored in the average range, 40th to 60th percentile, on the CAPE).

All participants were between the ages of 18 and 59 years, sufficiently fluent in Dutch, and without a history of neurological disorders such as epilepsy and concussion with loss of consciousness. Written informed consent, in accordance with the local ethical committee guidelines, was obtained from all participants.

Patients were recruited from the catchment area (source population: 350,000) for the Community Mental Health Centre and the catchment area for the Psychiatric Hospital. Initial inclusion criteria for patients were the lifetime prevalence of a period of psychosis (at least 2 weeks) in clear consciousness, according to the RDC (Research Diagnosis Criteria⁴⁴).

Relatives (free from a lifetime history of psychosis) were sampled through participating patients or through associations for relatives of patients with psychotic symptoms.

Participants with average and high levels of psychotic experiences were recruited from an earlier longitudinal family study in the general population conducted in the city of Sittard (Continuum of Mental Disorders study, COMED).⁴⁵ Participants of the COMED study were aged 36–65 years and had been randomly selected and sent a letter in which they were asked to participate. Additionally, participants were asked through a snowball-sampling procedure to invite their family members to participate. Overall, 768 participants from a total of 116 families were included. All participants filled in the CAPE.^{42,43} The participants with a mean (ie, between 40th and 60th percentile) and a high (ie, above the 75th percentile) score on the CAPE positive psychosis dimensions were invited to participate in the CoP study.

The present study included 45 patients with psychosis (39.5% inpatients), 47 nonpsychotic first-degree relatives, 41 participants with a high level of subclinical psychotic experiences (psychosis-prone) and 54 healthy controls with an average level of psychotic experiences (controls). Of the 47 healthy relatives, there were 13 mothers, 8 fathers, 15 sisters, 8 brothers, 2 daughters, and 1 son. Twenty-seven families contributed at least 1 patient and 1 relative. Four relatives participated without their ill family member.

All participants were screened for symptoms listed in the Operational Criteria Checklist for Psychotic Illness (OCCPI).⁴⁶ Where necessary, additional information was derived from interviews with ward staff or personal case managers. Using the combined information in the OCCPI, the computerized program OPCRIT⁴⁶ yielded RDC diagnoses.⁴⁴

Current use of illicit drugs and alcohol was assessed using section I of the Composite International Diagnostic Interview (CIDI, version 1.1).⁴⁷

Instruments

CAPE. The Community Assessment of Psychic Experiences⁴² (<http://www.cape42.homestead.com/>) is a self-report instrument developed to assess dimensions of subclinical psychosis phenotype. It includes dimensions of positive psychotic experiences (20 items), negative psychotic experiences (14 items), and depressive experiences (8 items). (A detailed description of the development of the CAPE can be found in several sources.^{42,43,48,49}) The CAPE was used to split up the psychosis-prone group from the control group in the general population sample.

SAPS and SANS. The Scale for the Assessment of Positive Symptoms (SAPS)⁵⁰ and the Scale for the Assessment of Negative Symptoms (SANS)^{51,52} are complementary instruments used to assess the severity of symptoms in patients with schizophrenia or other psychotic disorders. The goal of the SAPS is to assess positive symptoms and disorganization. The SANS is designed to rate the presence and severity of negative symptoms.

The SAPS contains 30 items divided over 4 domains: hallucinations, delusions, disorganization or bizarre behavior, and formal thought disorder. The SANS contains 20 items divided over 5 domains: affective flattening and blunting, alogia, avolition-apathy, anhedonia-asociality, and attentional impairment. In addition to the individual SAPS and SANS items, a global severity rating is made for each domain. Ratings were made by trained interviewers on the basis of a standard clinical interview, observed behavior during the interview, and review of all available clinical material. A subscale score for each domain was constructed as the sum of the scores for each item in that domain. The time frame covered by the rating was lifetime.

PSE. The purpose of the Present State Examination (PSE)⁵³ is to assess the presence and severity of symptoms associated with a broad range of major psychiatric disorders over a designated period (ie, last week) by means of a structured clinical interview with the patient. In this study only the sections that cover items signs and symptoms of delusions were used (29 items: PSE55–PSE59 and PSE71–PSE92, in addition to their subscale scores).

CDSS. The Calgary Depression Scale for Schizophrenia (CDSS) was developed to assess symptoms of major depressive disorder in patients with schizophrenia. The CDSS consists of 9 items, all of them typical depressive symptoms that do not appear to overlap with the negative symptoms of schizophrenia. The CDSS is a reliable and

valid measure of the severity of depressive symptoms in patients with schizophrenia.^{54–56}

The SAPS, SANS, and CDSS are developed for use in patients with psychotic disorders. The PSE was designed to provide dimensional ratings of symptoms and syndromes that are not wedded to any single classification or diagnostic system.

In the SAPS, as well as in the PSE, more than 10 different types of delusions, corresponding to their content, can be rated in severity on a 6-point or 4-point Likert scale respectively. The rationale for using these scales not only in patients but also in relatives and psychosis-prone participants in the general population is that, at the lower end of the spectrum, ratings like “supposed” and “mild” are used. This makes sensitivity sufficient to assess subclinical experiences.

General Intelligence. General intelligence was measured by a combined score on 1 performance subtest and 1 verbal subtest from the Groningen Intelligence Test (GIT), a widely used Dutch intelligence test.⁵⁷ This test yields results that are comparable to those of the Wechsler Adult Intelligence Scale–Revised.⁵⁸

General intelligence is lower in people with schizophrenia. Hence, an association between JTC and general intelligence could indicate that general intelligence has the role of a confounder or mediator between JTC and psychosis liability.

Beads Task. A computerized version of the beads task outlined by Phillips and Edwards⁵⁹ was used. In this experiment participants are shown a pair of jars on a screen. One jar contains 85 green and 15 red beads, and the other jar has the opposite ratio of green and red beads. Participants are informed of the proportions, and the jars are removed from view. One of the jars then is chosen, still hidden from view, and a bead is drawn from it and shown on the screen to the participant. Beads are sequentially being drawn and always replaced. Although the participants are told that beads are being selected randomly, the sequence of colors is predetermined according to the ratio of the 2 colors. The participant’s task is to work out whether the experimenter is drawing from the mainly green or the mainly red jar. In this study the condition is chosen in which participants are free to determine how many beads are drawn, and the trial is only terminated once they affirm that they are certain about their choice.

Analyses

Statistical analyses were carried out with STATA version 8.⁶⁰ A 4-level group variable was constructed reflecting the hypothesized order in liability for psychosis, with controls (coded 0), psychosis-prone participants (coded 1), relatives (coded 2), and patients (coded 3) in the highest category. The number of beads requested by a subject

yielded a continuous outcome variable within a range from 0 to 20. This variable was found to be non-normally distributed because the majority of participants requested less than 5 beads. In order to examine the JTC outcome, a variable was constructed indicating whether a subject showed a JTC reasoning bias, defined as requesting only 1 bead before deciding (hereinafter, JTC#1). This cutoff was chosen a priori as it reflects the most definite expression of the reasoning bias under investigation and should therefore be most discriminating between groups. In order to test this assumption, associations were also tested using less stringent cutoff values (ie, using 2, 3, or more beads before deciding).

JTC#1 and Psychosis Liability. The association between JTC#1 and psychosis liability (ie, the 4-level group variable reflecting liability for schizophrenia) was examined using logistic regression analysis, and progressively less stringent selections of JTC cutoffs were also considered. Effect sizes were expressed as odds ratios with their 95% confidence intervals. The following a priori selected confounders of the association between JTC and psychosis liability were included in the logistic regression model: age, sex, general intelligence, level of education (8-point scale, for the analysis summarized into 3 levels: low, medium, high), and use of psychotropic medication on a regular basis (self-reported as daily to weekly use versus less than daily to weekly use) and illicit drugs (self-reported as “present use” versus no use) or alcohol (self-reported as more than 20 units/week versus less than 20 units/week).

JTC#1 and Symptoms of Psychosis. The distribution of the subscale scores on the SAPS, SANS, and PSE was highly skewed because most participants were in partial or full remission or simply did not reach a level of psychotic symptoms that could be detected by these measures. Therefore, the subscale scores were dichotomized (score 0 or 1), a score of 0 indicating that the subject did not have any symptom on that domain, a score of 1 indicating that the subject had at least 1 of the symptoms in that domain. For those having any delusional symptoms, the sum of the (nondichotomized) subscale score of the delusions domains of the PSE was taken as a measure for the severity of the delusions. The distribution of these non-zero scores was normal, allowing the distribution to be divided by its tertiles creating 3 tertile groups. The distribution of the sum scores on the CDSS was moderately skewed, also allowing it to be divided by its tertiles, creating 3 tertile groups.

Associations between JTC#1 on the beads task and presence of delusions as measured with the PSE were examined using logistic regression analysis. For each significant association between JTC#1 and the *presence* of delusions, the association with *severity* was also assessed.

In order to assess whether any association with delusions was independent from other positive and negative

or depressive symptoms, subsequent analyses were performed in which all symptom domains, assessed with the SAPS, the SANS, and the CDSS, were entered simultaneously in the model.

In order to control for any effect of patient status, the association between JTC#1 and delusions was additionally adjusted for psychosis liability.

Any association between JTC#1 on the one hand and both the presence of psychotic symptoms, as well as psychosis liability, on the other was further examined by comparing the associations between JTC#1 and psychosis liability in a selection of participants with delusions with the associations in participants without delusions within each of the 3 groups.

In order to control for any bias induced by clinical severity, we examined the association between JTC and psychosis liability, as well as the association between JTC and delusions, when inpatients were excluded from the sample.

Furthermore, we examined whether any JTC reasoning bias was particularly associated with schizophrenia liability by excluding all patients with RDC diagnoses other than narrow schizophrenia and schizoaffective disorder.

Power Analysis. Assuming a proportion of 20% of JTC in the whole study sample and a relative risk of 3.5 in the patient group, the power to detect differences between the patient group and the control group at a conventional alpha-level of 0.05 (2-sided) is 0.8.

Results

Sample Characteristics

Two patients were excluded because data on diagnosis and symptoms were missing. Data on performance on the beads task were missing for 3 patients (all 3 with diagnosis of narrow schizophrenia), 7 relatives, and 1 subject from the control group, leaving 40 patients, 40 relatives, 41 psychosis-prone participants, and 53 controls. There were 28 patients (70.0 %) with a diagnosis of schizophrenia, 5 patients (12.5 %) with a diagnosis of schizoaffective disorder, and 5 patients (12.5 %) with a diagnosis of unspecified functional psychosis. For 2 patients (5.0%) OCCPI data were missing, but lifetime presence of positive psychotic symptoms was confirmed with the SAPS. The mean age of the whole sample was 41.6 years (SD = 11.2). The mean age of the patients was lower than that of the other groups (see Table 1). The patient group had a significantly lower general intelligence and level of education compared with the control group.

Antipsychotic agents were used in the past week by 28 participants, all of whom were in the patient group (28 of 40 patients, 70%). Eight out of 40 patients (20%) reported current use of illicit drugs, which in all cases but 2 was

Table 1. Summary Statistics of Participant Characteristics

	Controls (<i>n</i> = 53)		Psychosis prone (<i>n</i> = 40)		Relatives (<i>n</i> = 41)		Patients (<i>n</i> = 40)		F (<i>df</i> = 3)	<i>p</i>	Scheffé
	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
Age	47.4	7.4	44.3	9.8	40.1	11.9	33.3	10.3	17.15	.000	3 < 2, 1, 0; 2 < 0
Sex (male/female)	20/33		16/25		15/25		32/8		19.95 ^a	.000	
Educational level ^b	5.57	0.84	4.98	1.39	5.19	1.38	4.59	1.19	5.50	.001	3 < 0
Intelligence ^c	7.38	1.59	6.85	1.41	6.99	1.94	6.18	2.12	3.07	.029	3 < 0
Scale for the Assessment of Positive Symptoms (SAPS) ^d	0.09	0.35	0.37	0.97	0.53	1.37	5.33	4.17			
SAPS hallucinations	0	0	0.54	1.58	0.58	1.92	6.72	7.6			
SAPS delusions	0	0	0.34	1.88	0.63	2.2	11.23	8.6			
SAPS bizarre behavior	0.94	2.10	1.41	2.47	1.33	2.35	5.48	5.19			
SAPS thought disorder	0.04	0.19	0.58	1.30	0.13	0.46	2.15	3.65			
Scale for the Assessment of Negative Symptoms (SANS) ^e	0.19	0.56	0.15	0.53	0.40	1.26	2.63	3.53			
SANS apathy	0.19	0.62	0.27	0.70	0.7	2.19	2.92	4.06			
SANS anhedonia	1.13	1.71	1.10	1.88	0.86	1.68	3.87	5.00			
SANS affective flattening	0.11	0.61	0.21	1.04	0.20	0.76	4.82	7.77			
SANS alogia	0	0	0	0	0	0	0.77	2.00			
SANS attention	0	0	0.29	0.90	0.40	1.05	1.18	1.96			
Calgary Depression Scale for Schizophrenia (CDSS) ^f	1.34	2.68	2.83	3.85	1.67	2.88	2.88	3.51			

^aChi-square test.^bEducation was measured on an 8-point scale (primary school to university degree).⁹⁰^cIntelligence was measured on a 10-point scale derived from 2 subtests of the Groningen Intelligence Test.^dSAPS summary score (range 0–20).^eSANS summary score (range 0–25).^fCDSS total score (range 0–27).

restricted to cannabis. Two out of 53 controls (3.7%), 3 out of 41 delusion-prone participants (7.3%), and 5 out of 40 relatives (12.5%) reported use of illicit drugs, which in all cases but 1 (relative) was restricted to cannabis.

There was no significant difference in alcohol use between groups. There were 7 out of 53 controls (13.2%), 7 out of 41 delusion-prone participants (17.1%), 2 out of 40 relatives (5%), and 5 out of 40 patients (12.5%) who reported the use of more than 20 units alcohol/week.

Association Between JTC and Psychosis Liability

In the whole sample 35 (20.1%) of the participants showed a JTC#1 reasoning bias: 6 of 53 participants in the control group (11.3%), 6 of 41 in the psychosis-prone group (14.6%), 10 of 40 in the relatives (25%), and 13 of 40 in the patients (32.5%).

A linear trend was apparent in the association between JTC#1 and psychosis liability, the association being stronger as the psychosis liability increased (odds ratio [OR] linear trend = 1.59, 95% CI: 1.13–2.24). When entered as 3 dummy variables comparing associations with the reference control group, it reached significance for the patient group (OR = 3.77, 95% CI: 1.28–11.07; see

Table 2), indicating that the patient group was more likely to jump to conclusions than the controls. When the inpatients were excluded, the association between JTC and psychosis liability in the patient group reduced but did not nullify (OR = 3.23, 95% CI: 0.94–10.96). When only the patients with RDC diagnoses of narrow schizophrenia and schizoaffective disorder were included, the association between JTC and psychosis liability grew stronger and more specific (OR = 5.09, 95% CI: 1.70–15.29).

The strength and statistical precision of the association between JTC#1 and the psychosis liability variable was reduced when adjusted for the confounders age, gender, level of education, general intelligence, and use of cannabis or other illicit drugs (see Table 2). This reduction was largely attributable to the effect of “general intelligence.”

As the cutoff criterion of number of beads used to define JTC became progressively less stringent, the association between this selection and the schizophrenia liability variable was also progressively weaker, as expected (for JTC ≤ 2, in the patient group, OR = 2.23, 95% CI: 0.97–5.15; in the relatives, OR = 1.49, 95% CI: 0.65–3.43; and in the psychosis-prone group, OR = 0.95, 95% CI: 0.41–2.21).

Table 2. Associations Between Number of Beads Requested = 1 (JTC#1) and the Group Variable Reflecting Psychosis Liability (Linear Trend and Each Value Compared With Control Group)

JTC#1	Odds Ratio	<i>p</i>	95% CI
PL (linear trend) (unadjusted)	1.59	.008	1.13–2.24
PL (linear trend) ^a	1.57	.034	1.04–2.39
PL (linear trend) ^b	1.37	.10	0.94–1.99
PL (linear trend) ^c	1.34	.20	0.86–2.09
Group = 1	1.34	.63	0.40–4.52
Group = 2	2.61	.09	0.86–7.93
Group = 3	3.77	.016	1.28–11.07
PL; Group = 1 ^a	1.21	.76	0.35–4.23
PL; Group = 2 ^a	2.30	.16	0.72–7.35
PL; Group = 3 ^a	3.60	.06	0.96–13.44
PL; Group = 1 ^b	1.24	.73	0.36–4.23
PL; Group = 2 ^b	2.08	.21	0.66–6.55
PL; Group = 3 ^b	2.40	.15	0.73–7.92
PL; Group = 1 ^c	1.19	.78	0.34–4.14
PL; Group = 2 ^c	1.78	.35	0.53–5.94
PL; Group = 3 ^c	2.30	.25	0.56–9.50

Note: PL = psychosis liability (Group = 0: controls; Group = 1: psychosis prone; Group = 2: relatives; Group = 3: patients).

^aAdjusted for age, gender, level of education, and use of cannabis and other illicit drugs.

^bAdjusted for general intelligence.

^cAdjusted for age, gender, level of education, use of cannabis and other illicit drugs, and general intelligence.

Association Between JTC and Delusions

There was a significant association between JTC#1 and the dichotomized score on the delusions subscales from the PSE (OR = 2.59, 95% CI: 1.18–5.69). The association became less specific after adjustment for age, gender, level of education, general intelligence, and use of cannabis and other illicit drugs (OR = 6.72; 95% CI: 0.45–100.41). Again, the reduction in specificity was largely attributable to the effect of general intelligence.

When the inpatients were excluded from the sample, the association between JTC and delusions hardly changed (OR = 2.07, 95% CI: 0.80–5.29).

When the patients with RDC diagnoses other than narrow schizophrenia and schizoaffective disorder were excluded, the association between JTC and delusions grew stronger and more specific (OR = 3.13, 95% CI: 1.40–7.00).

As the cutoff criterion of number of beads used to define JTC became progressively less stringent, the association between this selection and the dichotomized score on the delusions subscales from the PSE was also progressively weaker.

In order to examine any dose-response relationship between JTC#1 and severity of delusions, the distribution of the non-zero scores on the delusions domain of the PSE was divided by its tertiles. Compared with those

Table 3. Logistic Regression With Number of Beads Requested and Level of Delusions Measured With the Present State Examination (PSE) (Each of the Tertiles of the Scores > 0 Compared With No Delusions)

JTC and PSE Delusions (tertiles)	JTC#1		
	Odds Ratio	<i>p</i>	95% CI
Linear trend	1.69	.004	1.18–2.43
1st tertile	1.43	.560	0.43–4.73
2nd tertile	2.86	.116	0.77–10.56
3rd tertile	5.00	.010	1.48–16.88

Note: Unadjusted odds ratios.

without delusions, the strength of the association increased with increasing levels of delusions, yielding a dose-response relationship (OR linear trend = 1.69, 95% CI: 1.18–2.43; Table 3). When entered as 3 dummy variables for comparison with the reference category of no delusions, it reached significance for the delusions score in the highest tertile, even when adjusted for age, gender, level of education, and use of cannabis and other illicit drugs (OR = 4.95, 95% CI: 1.20–20.33). The association was reduced but not nullified when general intelligence was added to the equation (OR = 3.49, 95% CI: 0.74–16.42).

Examining the association of each of the symptom domains of the SAPS and SANS and the scores on the CDSS with JTC#1, only the association with the dichotomized score on the delusions subscale proved significant (Table 4). After including all the subscale scores from the SAPS, SANS, and CDSS in 1 multiple logistic regression model, the association with this delusion subscale score was the strongest by far, although not significant anymore (OR = 3.07, 95% CI: 0.88–10.75).

Association Between JTC and Delusions and Psychosis Liability

The association between JTC#1 and the presence of delusions diminished when psychosis liability was brought into the equation (JTC#1 and presence of delusions, OR = 1.64, 95% CI: 0.59–4.54; JTC#1 and psychosis liability: OR = 1.4, 95% CI: 0.91–2.15).

Within each of the 3 groups, the association with JTC#1 in participants without delusions was compared with the association with JTC#1 in participants with delusions. In the patient group (coded 3) the association with JTC#1 was stronger in the participants with delusions (OR = 4.95, 95% CI: 1.62–15.09) than in those without (OR = 0.98, 95% CI: 0.10–9.25). In both at-risk groups, the associations with JTC#1 were not significant.

Association Between JTC and General Intelligence

There was a significant association between JTC#1 and general intelligence (OR = 0.79, 95% CI: 0.64–0.98).

Table 4. Associations Between JTC = 1 and Dichotomized Scores on All Symptom Domains of the SAPS and SANS and Depression on the CDSS (Tertiles) (Logistic Regression)

JTC#1 and SAPS/SANS subscales, CDSS	Odds ratio ^a	<i>p</i> ^a	95% CI ^a	Odds ratio ^b	<i>p</i> ^b	95% CI ^b
Hallucinations	2.04	0.092	0.89–4.68	0.84	0.772	0.26–2.71
Delusions	2.81	0.011	1.27–6.23	3.07	0.079	0.88–10.75
Disorganization	1.31	0.469	0.63–2.73	0.81	0.633	0.33–1.95
Formal thought disorder	2.33	0.057	0.98–5.55	1.65	0.329	0.60–4.52
Apathy	1.50	0.328	0.67–3.37	0.90	0.848	0.31–2.62
Anhedonia	1.42	0.345	0.68–2.97	1.14	0.759	0.50–2.56
Affective flattening	0.97	0.964	0.34–2.81	0.54	0.390	0.13–2.19
Alogia	1.12	0.886	0.22–5.67	0.59	0.612	0.08–4.50
Attention	2.14	0.096	0.87–5.24	2.03	0.209	0.67–6.11
Depression (tertiles)	1.26	0.318	0.80–1.98	1.09	0.724	0.65–1.85

Note: SAPS = Scale for the Assessment of Positive Symptoms; SANS = Scale for the Assessment of Negative Symptoms; CDSS = Calgary Depression Scale for Schizophrenia.

^aCalculated in different models with JTC and 1 symptom domain at a time.

^bCalculated in 1 equation with JTC and all symptom domains.

Discussion

The results show that there is a dose-response relationship in the association between JTC and psychosis liability. Controlling for age, gender, educational level, and drugs only reduced the association by small amounts. Adjusting for general intelligence generally reduced the association and made it no longer statistically significant. There was also a significant association between JTC reasoning bias and having delusions, and a dose-response relationship between JTC#1—ie, showing the highest possible and a priori hypothesized JTC reasoning bias—and the severity of delusions, even when adjusted for age, gender, and level of education. The association with delusions overlapped with the effect of psychosis liability. However, there was an interaction between psychosis liability and having delusions in their effect on JTC: no association with JTC#1 was found for the patients without delusions, whereas the association was strong and significant in those with delusions, indicating that the association with delusions cannot be attributed fully to the effect of having a diagnosis of psychosis.

Dimensions in Schizophrenia and JTC

When the associations between JTC#1 and each of the symptom domains of the SAPS/SANS and CDSS were considered independently, the association with delusions was the strongest, followed in order by the association with positive formal thought disorder, hallucinations, attention, and depression. When controlled for each other in 1 equation, this ranking did not change much, except for the association between JTC#1 and hallucinations, which became much weaker, suggesting that the association with hallucinations was indirectly occasioned by the strong association between hallucinations and

delusions.⁶¹ The association with the typical negative symptom domains (alogia, anhedonia, apathy, affective flattening) and behavioral disorganization remained the weakest.

The heterogeneous phenotype of schizophrenia has proven to be composed of separable, though correlated, symptom factors that can also be observed in their relatives and participants with schizotypy, according to most factor analytic studies. Typically, a positive and a negative symptom factor is found, in addition to a disorganization or a depressive factor.^{24,27–29,37,38,42,45,62–65} The findings in our study indicate that JTC is associated with the positive symptoms of delusions specifically and, to a much lesser extent and statistically imprecise, with other positive symptoms (such as hallucinations) and with components of the disorganizational symptom factor (attention, formal thought disorder).

The role of general intelligence and cognitive capacities in reasoning bias in delusions is poorly investigated. In other studies no evidence was found for a role of memory impairment,¹ and mixed evidence was found for the ability to process sequential information.^{3,4,66} In our study we found a significant association between JTC#1 and general intelligence. General intelligence impacted on the association between JTC and schizophrenia by reducing its effect size and the statistical precision. However, this does not mean that JTC cannot be causally implicated in the cognitive liability to psychosis. Rather, the current findings may indicate that JTC and general intelligence are both independently associated with schizophrenia or, alternatively, that JTC is a mediator in the association between general intelligence and schizophrenia. For example, a lower level of intelligence may lead to a jumping-to-conclusions cognitive style, which in turn may make someone vulnerable to develop delusional ideation.

Trait or State

The dose-response association between JTC and psychosis liability is in favor of the hypothesis that JTC has—at least in part—features of a trait reflecting liability for schizophrenia. The dose-response association between JTC and delusions indicates that JTC also has characteristics of a state, as it covaries with the level of delusions. The JTC reasoning bias could therefore be involved in the formation, as well as in the maintenance, of delusions.

It is a well-established finding that relatives of patients with schizophrenia are at higher risk for developing schizophrenia spectrum disorders and show more schizotypal signs and symptoms, which may be an attenuated expression of the trait.⁶⁷ The present findings, as well as previous studies, suggest that the psychological mechanisms associated with psychotic symptoms also seem to operate at lower levels of the continuum in these individuals.^{35,68–72} In previous studies some of the proposed mechanisms in delusion formation and/or maintenance were also found in relatives and delusion-prone individuals of the general population, notably the JTC reasoning bias^{3,4} and the attentional bias related to threatening social stimuli.^{70,71} Other mechanisms contributing to delusion formation and maintenance have features of a trait as well as a state—for example, in alterations in theory of mind^{68,73–75}—or only features of a state, as is the case with attributional bias.^{76,77} Apparently, psychological mechanisms associated with psychotic symptoms can have both state and traitlike features. However, the state-trait dichotomy in itself is somewhat problematic, and so is the inference that the difference between a trait and state determines whether a certain mechanism is involved in the formation or in the maintenance of delusions. First, a factor with a trait quality can be a necessary but not yet sufficient condition to develop a symptom, and a covarying state quality does not add much information with regard to etiological mechanisms. So there may be other factors in operation that make the trait come to expression, just like a (genetic) predisposition can come to expression under certain conditions.^{78–81} Second, as Bentall⁸² stated, the assumption behind the dichotomous trait-state distinction is that abnormalities are either present prior to the emergence of symptoms (in which case they may play a causal role) or covary with symptoms (in which case they may be either part of the symptom picture or epiphenomena). In fact, there is a larger range of possibilities in changes and interactions of qualities over time than just being stable or covarying together. It may be possible that one quality (for example, JTC) increases over time and at a certain point elicits the expression of another (for example, delusions). In order to clarify this relationship, a longitudinal design with more measurements in time would be more suitable. The dichotomy between state and trait therefore seems to be artificial, as it does not correspond to the way qualities

are present in nature, and provides little information on causal or even temporal relationships. Taking these remarks into account, it seems unlikely that a single factor such as a JTC reasoning bias can be linked with either the formation or the maintenance of delusional beliefs. More likely, a dynamic interplay exists between delusional symptoms and cognitive processes. For example, it is possible that the cognitive processes of deluded patients are not dysfunctional under optimum environmental conditions but, because of their reciprocal influences, are more easily “disturbed” by adverse events than those of individuals who never have delusional experiences.^{82,23}

Processes Involved in JTC

In recent paradigms of delusions, multifactorial models are proposed, in which changes in perceptual and cognitive processes, prior cultural conditioned beliefs, motivation, and affect may play a part.^{1,2,76,83}

The “jumping to conclusions” is in itself a complex phenomenon, in which different underlying cognitive processes may be involved. Both this and previous studies have suggested that people with delusions who jump to conclusions show a *data-gathering bias*, a tendency to seek less information to reach a decision.^{1,4,5} These individuals also show a *disconfirmation bias*: just as they are willing to accept a hypothesis on the basis of little evidence, they are also more ready to reject it on the basis of little potentially contradictory evidence.^{1,13,84} Furthermore, people with paranoid delusions also show a tendency to discard disconfirmatory evidence.⁸³ People with delusions and a JTC reasoning bias apparently do not show a *probabilistic reasoning bias*: their estimation of probabilities does not differ from other groups.^{1,4,5,84} Deluded patients also seemed to take random variation into account, in a variant of the beads task with base rate change (60:40 vs 85:15). When emotionally salient material was used, the JTC reasoning bias increased.^{6,13,85} Furthermore, people with delusions show a high need for closure: they prefer a definite answer compared with indecisiveness and ambiguity.^{86–88} Evidence is mixed, however, as to whether this need for closure is associated with JTC.^{3,7,88}

Finally, it could be possible that, in addition to a cognitive reasoning bias, other factors, such as impulse control, have an impact on the JTC bias. However, to our knowledge, no associations between impulse control and delusions or between impulse control and JTC have been reported. A previous study examining this issue concluded that the early responses do seem to reflect a reasoning bias, rather than impulsiveness.⁶

Limitations

Statistical power was restricted due to small numbers in some cases, especially when examining dose-response relationships. For example, only 14 individuals of those

jumping to conclusions after 1 bead had delusions, and in the relatives and high schizotypy group there were only 3 such individuals in each group.

Although subclinical phenotypes were investigated, we used psychometric instruments developed for clinical use in order to assess the presence and severity of delusions and other psychotic symptoms (SAPS/SANS/PSE). However, it has been shown in earlier work that clinical instruments such as the Brief Psychiatric Rating Scale (BPRS) can be suited for the assessment of subclinical phenomena in the general population.^{43,89}

JTC was associated with general intelligence. Controlling for general intelligence reduced the critical associations below the level of statistical significance. In order to determine the character of the relationship between general intelligence, JTC, and delusions, and in order to examine whether more specific cognitive domains, such as general reasoning, are responsible for the association with general intelligence, future studies using a broader range of cognitive tests should be carried out.

We assumed that requesting only 1 bead before deciding (JTC#1) reflects the most definite expression of the reasoning bias under investigation and should therefore be most discriminating. We tested this assumption only with the data of this very study, using less stringent cutoff values. To our knowledge, the noncontinuous JTC#1 has not been used or validated in any previous study. Since the beads task was developed and used in previous studies as more of a continuous measure, this can be regarded a psychometric limitation.

The data in this study come from a snapshot comparison between groups, lacking the variable “time,” which is useful in discriminating between a trait and state quality. For example, our study does not supply information on how changes in JTC and changes in delusions are related over time. Furthermore, although a measurement of psychotic symptoms during the last week, as well as lifetime, was available, only 4 participants reported having had delusions in their lifetime but not in the past week, which may suggest that participants were prone to report recent experience when asked for lifetime experiences.

In conclusion, we showed that there is a dose-response relationship between JTC and psychosis liability and between JTC and the severity of delusions, partly overlapping with the association between JTC and general intelligence. We argued that the manifestation of delusions is the result of a dynamic interplay between JTC and other factors. JTC seems correlated with the presence of delusions and, to a lesser extent, with other positive symptoms and with the disorganizational symptom factor of schizophrenia.

Further research with a similar but longitudinal design, in which more realistic situations, disconfirmation bias, need for closure, emotional salience, and metacognition are brought into the model, is needed for a better understanding of the formation and maintenance of delu-

sions, which may eventually lead to targeted cognitive-behavioral interventions.

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References

1. Garety PA, Freeman D. Cognitive approaches to delusions: a critical review of theories and evidence. *Br J Clin Psychol.* 1999;38:113–154.
2. Garety PA, Kuipers E, Fowler D, Freeman D, Bebbington PE. A cognitive model of the positive symptoms of psychosis. *Psychol Med.* 2001;31:189–195.
3. Colbert SM, Peters ER. Need for closure and jumping-to-conclusions in delusion-prone individuals. *J Nerv Ment Dis.* 2002;190:27–31.
4. Linney YM, Peters ER, Ayton P. Reasoning biases in delusion-prone individuals. *Br J Clin Psychol.* 1998;37:285–302.
5. Dudley RE, John CH, Young AW, Over DE. The effect of self-referent material on the reasoning of people with delusions. *Br J Clin Psychol.* 1997;36:575–584.
6. Dudley RE, John CH, Young AW, Over DE. Normal and abnormal reasoning in people with delusions. *Br J Clin Psychol.* 1997;36:243–258.
7. Dudley RE, Over DE. People with delusions jump to conclusions: a theoretical account of research findings on the reasoning of people with delusions. *Clin Psychol Psychother.* 2003;10:263–274.
8. Huq SF, Garety PA, Hemsley DR. Probabilistic judgements in deluded and non-deluded subjects. *Q J Exp Psychol A.* 1988;40:801–812.
9. John CH, Hodgson C. Inductive reasoning in delusional thought. *J Ment Health.* 1994;3:31–49.
10. Fear CF, Healy D. Probabilistic reasoning in obsessive-compulsive and delusional disorders. *Psychol Med.* 1997;27:199–208.
11. Mortimer AM, Bentham P, McKay AP, et al. Delusions in schizophrenia: a phenomenological and psychological exploration. *Cogn Neuropsychiatry.* 1996;1:289–303.
12. Peters ER, Day S, Garety P. From preconscious to conscious processing: where does the abnormality lie in delusions? *Schizophr Res.* 1997;24:120.
13. Young HF, Bentall RP. Probabilistic reasoning in deluded, depressed and normal subjects: effects of task difficulty and meaningful versus non-meaningful material. *Psychol Med.* 1997;27:455–465.
14. Conway CR, Bollini AM, Graham BG, Keefe RS, Schiffman SS, McEvoy JP. Sensory acuity and reasoning in delusional disorder. *Compr Psychiatry.* 2002;43:175–178.
15. Moritz S, Woodward TS. Jumping to conclusions in delusional and non-delusional schizophrenic patients. *Br J Clin Psychol.* 2005;44:193–207.
16. Peters ER, Garety PA. A longitudinal study of cognitive abnormalities in delusions at different levels of information processing. *Schizophr Res.* 1999;36:180.
17. Peters ER. Cognitive biases involved in the formation of delusional beliefs. *Schizophr Res.* 2003;60(Suppl 1):178.

18. Peters E, Garety P. Cognitive functioning in delusions: A longitudinal analysis. *Behav Res Ther.* 2005;in press. Epub ahead of print, May 2005.
19. Broome M, Brett C, Johns LC, et al. Reasoning styles and delusions in early psychosis. *Schizophr Res.* 2003;60(Suppl 1):12–13.
20. Dent J, Teasdale JD. Negative cognition and the persistence of depression. *J Abnorm Psychol.* 1988;97:29–34.
21. Brown GW, Bifulco A, Andrews B. Self-esteem and depression: IV. effect on course and recovery. *Soc Psychiatry Psychiatr Epidemiol.* 1990;25:244–249.
22. Brown GW, Andrews B, Harris T, Adler Z, Bridge L. Social support, self-esteem and depression. *Psychol Med.* 1986;16: 813–831.
23. van Os J, Jones P, Sham P, Bebbington P, Murray RM. Risk factors for onset and persistence of psychosis. *Soc Psychiatry Psychiatr Epidemiol.* 1998;33:596–605.
24. Fanous A, Gardner C, Walsh D, Kendler KS. Relationship between positive and negative symptoms of schizophrenia and schizotypal symptoms in nonpsychotic relatives. *Arch Gen Psychiatry.* 2001;58:669–673.
25. Baron M, Gruen R, Rainer JD, Kane J, Asnis L, Lord S. A family study of schizophrenic and normal control probands: implications for the spectrum concept of schizophrenia. *Am J Psychiatry.* 1985;142:447–455.
26. Kendler KS, McGuire M, Gruenberg AM, O'Hare A, Spellman M, Walsh D. The Roscommon Family Study: I. methods, diagnosis of probands, and risk of schizophrenia in relatives. *Arch Gen Psychiatry.* 1993;50:527–540.
27. Johns LC, van Os J. The continuity of psychotic experiences in the general population. *Clin Psychol Rev.* 2001;21: 1125–1141.
28. van Os J, Hanssen M, Bijl RV, Vollebergh W. Prevalence of psychotic disorder and community level of psychotic symptoms: an urban-rural comparison. *Arch Gen Psychiatry.* 2001;58:663–668.
29. van Os J, Hanssen M, Bijl RV, Ravelli A. Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophr Res.* 2000;45:11–20.
30. Tien AY. Distributions of hallucinations in the population. *Soc Psychiatry Psychiatr Epidemiol.* 1991;26:287–292.
31. Gilvarry CM, Russell A, Jones P, Sham P, Hemsley D, Murray RM. Verbal fluency in patients with schizophrenia and affective psychoses and their first-degree relatives. *Psychol Med.* 2001;31:695–704.
32. Keefe RS, Silverman JM, Roitman SE, et al. Performance of nonpsychotic relatives of schizophrenic patients on cognitive tests. *Psychiatry Res.* 1994;53:1–12.
33. Kremen WS, Seidman LJ, Pepple JR, Lyons MJ, Tsuang MT, Faraone SV. Neuropsychological risk indicators for schizophrenia: a review of family studies. *Schizophr Bull.* 1994;20:103–119.
34. Faraone SV, Kremen WS, Lyons MJ, Pepple JR, Seidman LJ, Tsuang MT. Diagnostic accuracy and linkage analysis: how useful are schizophrenia spectrum phenotypes? *Am J Psychiatry.* 1995;152:1286–1290.
35. Krabbendam L, Marcelis M, Delespaul P, Jolles J, van Os J. Single or multiple familial cognitive risk factors in schizophrenia? *Am J Med Genet.* 2001;105: 183–188.
36. Green MF, Nuechterlein KH. Backward masking performance as an indicator of vulnerability to schizophrenia. *Acta Psychiatr Scand Suppl.* 1999;395:34–40.
37. Faraone SV, Seidman LJ, Kremen WS, Toomey R, Pepple JR, Tsuang MT. Neuropsychologic functioning among the nonpsychotic relatives of schizophrenic patients: the effect of genetic loading. *Biol Psychiatry.* 2000;48:120–126.
38. Claridge G. Single indicator of risk for schizophrenia: probable fact or likely myth? *Schizophr Bull.* 1994;20:151–168.
39. Schurhoff F, Szoke A, Meary A, et al. Familial aggregation of delusional proneness in schizophrenia and bipolar pedigrees. *Am J Psychiatry.* 2003;160:1313–1319.
40. Whalley HC, Simonotto E, Flett S, et al. fMRI correlates of state and trait effects in subjects at genetically enhanced risk of schizophrenia. *Brain.* 2004;127:478–490.
41. Shedlack K, Lee G, Sakuma M, et al. Language processing and memory in ill and well siblings from multiplex families affected with schizophrenia. *Schizophr Res.* 1997; 25:43–52.
42. Stefanis NC, Hanssen M, Smirnis NK, et al. Evidence that three dimensions of psychosis have a distribution in the general population. *Psychol Med.* 2002;32:347–358.
43. Hanssen M, Krabbendam L, Vollema M, Delespaul P, van Os J. Evidence for instrument and family-specific variation of subclinical psychosis dimensions in the general population. *J Abnorm Psychol.* 2005;in press.
44. Spitzer RL, Endicott J, Robins E. Research diagnostic criteria: rationale and reliability. *Arch Gen Psychiatry.* 1978;35: 773–782.
45. Hanssen M, Peeters F, Krabbendam L, Radstake S, Verdoux H, van Os J. How psychotic are individuals with non-psychotic disorders? *Soc Psychiatry Psychiatr Epidemiol.* 2003;38:149–154.
46. McGuffin P, Farmer A, Harvey I. A polydiagnostic application of operational criteria in studies of psychotic illness: development and reliability of the OPCRIT system. *Arch Gen Psychiatry.* 1991;48:764–770.
47. Smeets RD. *Composite International Diagnostic Interview (CIDI) Versie 1.1.* Amsterdam and Geneva: World Health Organization; 1993.
48. Verdoux H, van Os J. Psychotic symptoms in non-clinical populations and the continuum of psychosis. *Schizophr Res.* 2002;54:59–65.
49. Hanssen MS, Bijl RV, Vollebergh W, van Os J. Self-reported psychotic experiences in the general population: a valid screening tool for DSM-III-R psychotic disorders? *Acta Psychiatr Scand.* 2003;107:369–377.
50. Andreasen NC. *Scale for the Assessment of Positive Symptoms (SAPS).* Iowa City: University of Iowa; 1984.
51. Andreasen N. *Scale for the Assessment of Negative Symptoms (SANS).* Iowa City: University of Iowa; 1983.
52. Andreasen NC. Negative symptoms in schizophrenia: definition and reliability. *Arch Gen Psychiatry.* 1982;39:784–788.
53. Wing JK, Cooper JE, Sartorius N. *Measurement and Classification of Psychiatric Symptoms: An Instruction Manual for the PSE and Catego Program.* London: Cambridge University Press; 1974.
54. Addington D, Addington J, Maticka-Tyndale E. Specificity of the Calgary Depression Scale for schizophrenics. *Schizophr Res.* 1994;11:239–244.
55. Addington D, Addington J, Maticka-Tyndale E. Rating depression in schizophrenia: a comparison of a self-report and an observer report scale. *J Nerv Ment Dis.* 1993;181:561–565.
56. Addington D, Addington J, Maticka-Tyndale E. Assessing depression in schizophrenia: the Calgary Depression Scale. *Br J Psychiatry Suppl.* 1993;39–44.

57. Luteijn F, van der Ploeg F. *Handleiding Groninger Intelligentietest (GIT)* [Manual Groningen Intelligence Test]. Lisse, The Netherlands: Swets and Zeitlinger; 1983.
58. Wechsler D. *Wechsler Adult Intelligence Scale—Revised*. New York: Psychological Corporation; 1981.
59. Phillips LD, Edwards W. Conservatism in a simple probabilistic inference task. *J Exp Psychol*. 1966;72:346–354.
60. StataCorporation. *STATA Statistical Software: Release 8.0*. College Station, Tex: Stata; 1984–2003.
61. Krabbendam L, Myin-Germeys I, Hanssen M, et al. Hallucinatory experiences and onset of psychotic disorder: evidence that the risk is mediated by delusion formation. *Acta Psychiatr Scand*. 2004;110:264–272.
62. Lenzenweger MF, Dworkin RH. The dimensions of schizophrenia phenomenology: not one or two, at least three, perhaps four. *Br J Psychiatry*. 1996;168:432–440.
63. Stuart GW, Pantelis C, Klimidis S, Minas IH. The three-syndrome model of schizophrenia: meta-analysis of an artefact. *Schizophr Res*. 1999;39:233–242.
64. Claridge G, Beech T. Fully and quasi-dimensional constructions of schizotypy. In: Raine AE, Lencz TE, Mednick SA, eds. *Schizotypal Personality*. New York: Cambridge University Press; 1995:192–216.
65. Claridge G, McCreery C, Mason O, et al. The factor structure of “schizotypal” traits: a large replication study. *Br J Clin Psychol*. 1996;35:103–115.
66. Young HF, Bentall RP. Hypothesis testing in patients with persecutory delusions: comparison with depressed and normal subjects. *Br J Clin Psychol*. 1995;34:353–369.
67. Kendler KS, McGuire M, Gruenberg AM, O’Hare A, Spellman M, Walsh D. The Roscommon Family Study: III. schizophrenia-related personality disorders in relatives. *Arch Gen Psychiatry*. 1993;50:781–788.
68. Janssen I, Krabbendam L, Jolles J, van Os J. Alterations in theory of mind in patients with schizophrenia and non-psychotic relatives. *Acta Psychiatr Scand*. 2003;108:110–117.
69. Green MJ, Phillips ML. Social threat perception and the evolution of paranoia. *Neurosci Biobehav Rev*. 2004;28:333–342.
70. Green MJ, Williams LM, Davidson DJ. Visual scanpaths and facial affect recognition in delusion-prone individuals: increased sensitivity to threat? *Cogn Neuropsychiatry*. 2003;8:19–41.
71. Green MJ, Williams LM, Davidson DJ. Processing of threat-related affect is delayed in delusion-prone individuals. *Br J Clin Psychol*. 2001;40:157–165.
72. van Os J, Verdoux H, Maurice-Tison S, et al. Self-reported psychosis-like symptoms and the continuum of psychosis. *Soc Psychiatry Psychiatr Epidemiol*. 1999;34:459–463.
73. Wykes T, Hamid S, Wagstaff K. Theory of mind and executive functions in the non-psychotic siblings of patients with schizophrenia. *Schizophr Res*. 2001;49(Suppl):148–149.
74. Corcoran R, Mercer G, Frith CD. Schizophrenia, symptomatology and social inference: investigating “theory of mind” in people with schizophrenia. *Schizophr Res*. 1995;17:5–13.
75. Frith CD, Corcoran R. Exploring “theory of mind” in people with schizophrenia. *Psychol Med*. 1996;26:521–530.
76. Bentall RP, Corcoran R, Howard R, Blackwood N, Kinderman P. Persecutory delusions: a review and theoretical integration. *Clin Psychol Rev*. 2001;21:1143–1192.
77. Bentall RP, Kinderman P, Kaney S. The self, attributional processes and abnormal beliefs: towards a model of persecutory delusions. *Behav Res Ther*. 1994;32:331–341.
78. van Os J, Marcelis M. The ecogenetics of schizophrenia: a review. *Schizophr Res*. 1998;32:127–135.
79. Myin-Germeys I, Krabbendam L, Delespaul PA, van Os J. Do life events have their effect on psychosis by influencing the emotional reactivity to daily life stress? *Psychol Med*. 2003;33:327–333.
80. Myin-Germeys I, Krabbendam L, Delespaul P, van Os J. Can cognitive deficits explain differential sensitivity to life events in psychosis? *Soc Psychiatry Psychiatr Epidemiol*. 2003;38:262–268.
81. Myin-Germeys I, van Os J, Schwartz JE, Stone AA, Delespaul PA. Emotional reactivity to daily life stress in psychosis. *Arch Gen Psychiatry*. 2001;58:1137–1144.
82. Bentall RP. Commentary on Garety and Freeman: III: three psychological investigators and an elephant. *Br J Clin Psychol*. 1999;38:323–327.
83. Freeman D, Garety PA, Kuipers E, Fowler D, Bebbington PE. A cognitive model of persecutory delusions. *Br J Clin Psychol*. 2002;41:331–347.
84. Garety PA, Hemsley DR, Wessely S. Reasoning in deluded schizophrenic and paranoid patients: biases in performance on a probabilistic inference task. *J Nerv Ment Dis*. 1991;179:194–201.
85. McGuire L, Junginger J, Adams SG Jr, Burright R, Donovick P. Delusions and delusional reasoning. *J Abnorm Psychol*. 2001;110:259–266.
86. Kruglanski AW. *Lay Epistemics and Human Knowledge: Cognitive and Motivational Bases*. New York: Plenum; 1989.
87. Kruglanski AW, Webster DM. Motivated closing of the mind: “seizing” and “freezing.” *Psychol Rev*. 1996;103:263–283.
88. Bentall RP, Swarbrick R. The best laid schemas of paranoid patients: autonomy, sociotropy and need for closure. *Psychol Psychother*. 2003;76:163–171.
89. Krabbendam L, Myin-Germeys I, Hanssen M, van Os J. Familial covariation of the subclinical psychosis phenotype and verbal fluency in the general population. *Schizophr Res*. 2005;74:37–41.
90. De Bie SE. *Standaardvragen 1987: Voorstellen voor uniformering van vraagstellingen naar achtergrondkenmerken en interviews* [Standard questions 1987: proposal for uniformization of questions regarding background variables and interviews]. 2d ed. Leiden, The Netherlands: Leiden University Press. 1987.